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1: Enzyme Protein. 1996;49(1-3):7-19. Related Articles, Links

MMP-2: expression, activation and inhibition.

Corcoran ML, Hewitt RE, Kleiner DE Jr, Stetler-Stevenson WG.

Extracellular Matrix Pathology Section, National Cancer Institute, National Institutes of Health, Bethesda, Md., USA.

Remodeling of the extracellular matrix (ECM), which occurs during many physiological and pathological processes, is one of the requisite events of cellular invasion. The matrix metalloproteinases (MMPs) are a family of zinc-dependent proteases that are responsible for proteolytic degradation of specific ECM components. Regulating the activity of the MMPs at both mRNA and/or protein levels modulates the degradation of the ECM components which in turn alter cellular invasion. Although most MMPs are regulated via similar mechanisms at the mRNA and protein levels, the modulation of gelatinase A is unique. Understanding the mechanisms that regulate gelatinase A is important since expression and activation of this particular MMP is consistently correlated with a majority of malignant phenotypes. In this report, we will contrast the mechanisms that regulate the expression, activation and inhibition of gelatinase A with the mechanisms that modulate the rest the MMP family.

Publication Types:
- Review
- Review, Tutorial

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low-to-moderate levels of TIMP2 promote the activation of MMP2, whereas higher levels inhibit its activation by saturating free MT-MMPs that are needed to remove the MMP2 prodomain (Strongin et al. 1995). TIMP2 protein levels are reduced and MMP2 activation is enhanced in the presence of the MMP2 substrate, type IV collagen (Maquoi et al. 2000). Furthermore, the ability of collagen to induce MMP2 activation on demand probably results from TIMP2 degradation because there are no accompanying changes in MMP2, MT1-MMP, or TIMP2 mRNA expression or in the synthesis or activation of MT1-MMP. Therefore, local accumulation of type IV collagen may trigger its own degradation by somehow lowering local TIMP2 concentrations to levels that favor MMP2 activation.

Endogenous Metalloproteinase Inhibitors

The TIMPs represent a family of at least four 20–29-kDa secreted proteins (TIMPs 1–4) that reversibly inhibit the MMPs in a 1:1 stoichiometric fashion (reviewed in Edwards 2001, Sternlicht & Werb 1999, Gomez et al. 1997). They share 37–51% overall sequence identity, a conserved gene structure, and 12 similarly separated cysteine residues. These invariant cysteines form six intrachain disulfide bridges to yield a conserved six-loop, two-domain structure. Truncated "tiny" TIMPs 1 and 2 retain their inhibitory activity despite containing only the first three loops, thus indicating that portions of the N-terminal domain interact with the MMP catalytic site (Murphy & Willenbrock 1995). Mutational analyses (O'Shea et al. 1992, Willenbrock & Murphy 1994, Huang et al. 1997) and peptide- and antibody-blocking experiments (Bodden et al. 1994) have helped to further specify which regions of the N-terminal domain influence inhibitory function. In addition, NMR (Williamson et al. 1997) and X-ray crystallographic studies (Gomis-Rüth et al. 1997) have revealed which TIMP residues interact directly with the MMP3 catalytic domain and how they inhibit MMP activity. Although these studies indicate that the inhibitory activity of the TIMPs resides almost entirely in the N-terminal domain alone, both domains influence enzyme-inhibitor binding (Willenbrock & Murphy 1994). For example, the C-terminal domain (loops 4–6) of TIMP1 binds the hemopexin domain of MMP9 more readily than it does the hemopexin domain of MMP2, whereas the C-terminal domain of TIMP2 preferentially binds the hemopexin domain of MMP2 (Murphy & Willenbrock 1995).

Individual TIMPs differ in their ability to inhibit various MMPs (reviewed in Woessner & Nagase 2000). For example, TIMP2 and TIMP3 inhibit MT1-MMP, whereas TIMP1 does not. Likewise, TIMP1 is a relatively poor inhibitor of MT3-MMP, and TIMP3 appears to be a more potent inhibitor of MMP9 than other TIMPs. TIMP3 is also unique in its ability to inhibit ADAMs-10 and -17, ADAMTS-4, and ADAMTS-5 (Kashiwagi et al. 2001), whereas TIMP1 can inhibit ADAMTS-1 (Tortorella et al. 1999). In addition, the TIMPs differ in terms of their gene regulation and tissue-specific patterns of gene expression (Edwards 2001). TIMP3 also has the unique ability to bind via its C-terminal domain to heparan sulfates proteoglycans within the ECM, thereby concentrating it to specific regions within tissues and basement membranes (Langton et al. 1998).